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Ethnicity as a variable may be too greatly influenced by cultural attitude and therefore biased. In the past, this attitude led to the entire invention of diseases on the basis of race. At a time when the genetic inequality of races was considered obvious, the existence of these diseases was not questioned. In the present, ethnicity may be used euphemistically to avoid racist implications. A survey of 48 medical schools in the United States revealed that up to 91% of clerkship directors answered “yes” or “variable” after being queried whether students were taught by example to use the terms “black” or “white” when introducing case presentations. In another study, “black” patients were far more likely than “whites” to be racially identified at morning report. As recently as 1991, “black” patients were far more likely than “whites” to be racially identified at morning report. As recently as 1991, “black” patients were far more likely than “whites” to be racially identified at morning report. As recently as 1991, “black” patients were far more likely than “whites” to be racially identified at morning report. As recently as 1991, “black” patients were far more likely than “whites” to be racially identified at morning report.

To avoid the shortcomings linked to classification, it has been proposed (and is now used in many reports) that patients entering population studies “self-classify” their ethnicity, assuming that “racial and ethnic categories are understood by everyone” is true. However, misinterpretation, confusion, and self-reclassification have been found in these cases. One striking example is that the category “South and Central American” was thought by respondents in one census to refer to natives of the south and central United States. To define disease on the basis of biologically determined ethnicity might perhaps hide socioeconomic differences that are the real cause of disease in some ethnic groups. Affluent “blacks” from the US East Coast may not necessarily develop the same disease patterns as poor inhabitants from a comparable group in the southern states. Many reports on interethnic differences are not adjusted for socioeconomic variables, and this is perhaps the more important bias involving ethnicity research.

Senior and Bhopal have proposed that a “sound epidemiological variable” should be accurately measured and should differentiate populations by some underlying characteristic relevant to health (such as income, childhood circumstances, hormonal status, genetic inheritance, and lifestyle).
The observed differences in patterns of disease should generate testable etiologic hypotheses or be applicable to the planning and delivery of health care. However, these observed differences have rarely led to applicable hypotheses beyond the initial observation, as researchers have emphasized the understanding of etiology rather than the development of specific health policies.

Because ethnicity is difficult to define, it is consequently difficult to measure. Ethnic populations are more and more heterogeneous, and ethnic heterogeneity is often underestimated. An example are the well-conducted studies of coronary heart disease and stroke in “Japanese” men living in Japan and in “Japanese-American” men from Hawaii and California (the Ni-Hon-San studies), which showed a gradient from high stroke rates in Japan to low rates in California and a reverse trend for coronary heart disease.28–30 More than 11 000 men were included. These theoretically conflicting trends could not be explained only by conventional risk factors31 or blood pressure distributions,32 “although the subjects shared a common ethnic background.”33 It is very probable that ethnic heterogeneity is as important a confounder to the findings as environmental, dietary, and various other factors. The highly intriguing results of such studies, rather than being dismissed, merit a more rigorous approach.

Genetic research may be one way of achieving this. For instance, in the late eighties, familial cavernous malformations were reported to be more common among “Hispanics.”34,35 However, more precise research showed linkage of these cavernous malformations to chromosome 7q11–22 in 4 unrelated Mexican families that might share a common ancestor in Sonora County, Mexico.36 We now also know that “white”37 and “Italian-American”38 families with cavernous malformations may show the same or other linkages, for instance, to chromosomes 7p13–15 and 3q25.2–27.39,40 Of course, the familial occurrence points to an autosomal dominant, and a highly probable genetic linkage, but all the same, this is an example of how a bit more precision can help us avoid inappropriate, incorrect, or even suspect generalizations.

The consequences of flawed ethnicity research may lead to the assumption that ethnic minorities are an unhealthy social burden, that there are “ethnic” diseases which separate specific groups from the general population, that consequently they do not merit any further attention, and that “whites” are the “gold standard” of health. All this could do nothing but fuel racial prejudice.15 Patterns of disease that distinguish different ethnic groups may well exist and should be adequately established if better health standards for the populations concerned can be achieved as a consequence. Failure to do so may just be an example of “black box epidemiology,” resulting in futile and never-ending reports on interethnic differences that have been likened to repeatedly punching a soft pillow.41

References


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Ethnicity and Stroke: Beware of the Fallacies
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